A Case of Ileus and ST Segment Elevation

OSAMA SIDDIQUE, MD; SOMWAIL RASLA, MD; SETH CLARK, MD; ARAVIND KOKKIRALA, MD

ABSTRACT

ST segment elevation is associated with non-cardiac pathologies but is not as well reported as myocardial infarction. We present a case of a 63-year-old man who was admitted for an overdose on cyclobenzaprine with signs of anti-cholinergic toxicity. He developed signs of ileus on imaging and became progressively obtunded. He was noted to have ST segment elevations on electrocardiogram (EKG) with no troponin elevation. Patient required urgent catheterization which showed normal coronary arteries. His bowel was decompressed subsequently resulting in resolution of the ST segment changes. Other cases of ST segment elevations with gastrointestinal pathologies including cholecystitis, pancreatitis and gastric dilation have been reported but the etiology is still unclear. This case illustrates the importance of understanding EKGs in the clinical context. ST segment elevation on EKG, if there is contradicting symptomology and lab reports, should be further investigated to prevent unnecessary work-up and potentially dangerous therapies.

KEYWORDS: Ileus, non-cardiac, ST segment elevation, STEMI

INTRODUCTION

ST segment elevation is one of the widely used indicators of underlying significant myocardial infarction. Timely recognition and actions, including cardiac catheterization, are critical in improving outcomes in ST segment elevation myocardial infarction.

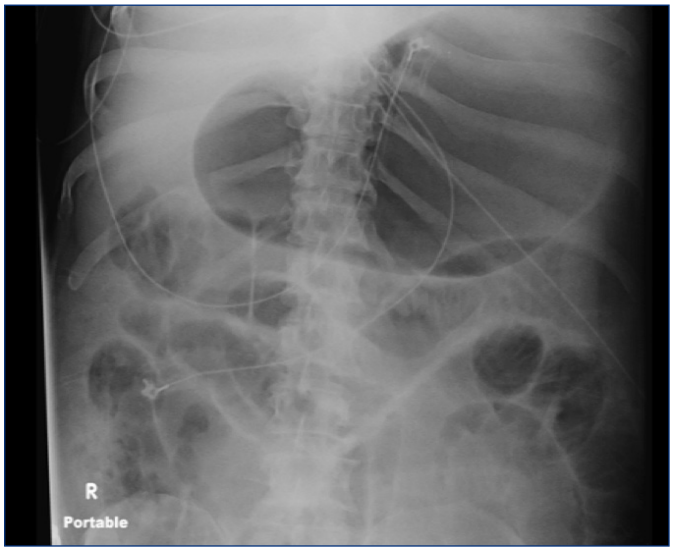
However, ST segment elevation has been associated not only with additional cardiovascular pathologies including: pericarditis[1], myocarditis[2], aortic dissection[3], takotsubo cardiomyopathy and pulmonary embolism[4] but also gastrointestinal etiologies such as gastric dilation[5], acute cholecystitis[6], and pancreatitis[7]. Therefore, a comprehensive differential for ST segment elevation should be kept in mind to avoid delay in time-sensitive investigations as well as unnecessary complications of thrombolysis and angioplasty. We report a case of ileus associated with ST segment elevation mimicking myocardial infarction (MI).

CASE REPORT

A 63-year-old man was referred for psychiatric evaluation. His past medical history was significant for hypertension, hyperlipidemia, obstructive sleep apnea, alcoholism, depression and a remote history of smoking. The patient had initially presented to another hospital due to overdose on Meloxicam, Ibuprofen and Cyclobenzaprine. He was admitted to a step-down unit and monitored for three days. His delirium improved and he started to regain bowel movements, which remained irregular, alternating between constipation and diarrhea. Paroxysmal atrial fibrillation developed, which was controlled by an oral calcium channel blocker. He was not started on anticoagulation given his low stroke risk with Congestive heart failure, Hypetension, Age, Diabetes, History of stroke, Sex, History of vascular disease (CHA_2DS_2-VASc) score of one.

On admission the patient was in stable condition but did not move his bowel on the first admission day. This was thought to be due to the remaining effect of recent anticholinergic toxicity associated with Cyclobenzaprine. His temperature (Temp) was 98.4, heart rate (HR) 95, blood pressure (BP) 140/90 and oxygen saturation (O2 Sat) 98% on room air. His electrocardiogram (EKG) at baseline showed sinus rhythm with no ST changes. The patient started to have nausea and dry heaves on the morning of the second day

Image 1. Abdominal X-ray (Kidney, Ureter, Bladder film) showing a large air density in the gastric body and antrum.



of admission. He denied any chest pain but complained of shortness of breath. His mental status progressed to an agitated delirium with disorientation. Temp was 98, HR 130, BP 110/70, O2 Sat 96% on ventilator. On examination he had new-onset abdominal distention with diffuse tympani. An abdominal X-ray showed pattern consistent with ileus (**Image 1**). A surgery consult was requested for suspicion of acute abdomen vs small intestinal obstruction. Simultaneous work-up for other causes of acute change in his mental status was sent including: liver function test, blood ammonia level, serum electrolytes, and Troponin-I, but all the results came back within the normal reference range, including Troponin of <0.01 (reference range 0.01-0.03). The patient's mental status declined further requiring emergent intubation. The EKG ordered with the blood work showed ST segment elevation on the inferior leads II, III and AVF, and V4-V6 which were new compared to the EKG on admission (**Image 2**). Cardiology was consulted and the patient was urgently transferred for a left heart catheterization (LHC) which demonstrated no signs of significant obstructive coronary lesions (**Image 3**). Although an emergent bedside echocardiogram had limited views prior to the LHC, it showed normal left ventricular function and no apical ballooning. The patient's cardiac enzymes remained negative after his cardiac angiography. His follow-up EKG (**Image 4**) obtained right after the decompression of his ileus showed resolution of his ST segments elevations.

DISCUSSION

ST segment elevation is considered one of the main features clinicians look for on an EKG to identify underlying acute ischemia. Many non-ischemic etiologies have a shared pattern of ST segment elevations, mainly pericarditis, myocarditis, aortic dissection and prinzmetal angina[8].

Non-cardiac etiologies have previously been reported as a cause of ST segment elevation. Chen et al. reported intraoperative pseudo myocardial infarction during a case of esophageal reconstruction where they conjectured that compression of the heart created an injury pattern in the inferior leads and clinical symptom of chest pain that had been mistaken as an acute myocardial infarction[9]. Moreover, a report of acute pancreatitis associated with ST segment elevation suggested that changes in the vagal nervous systems may be the culprit for the EKG changes[10]. Acute cholecystitis has also been associated with ST segment elevations despite negative echocardiography and angiography

Image 2. Sinus tachycardia, rate 126, ST segment elevations on Lead II, III, AVF and V4-V6.

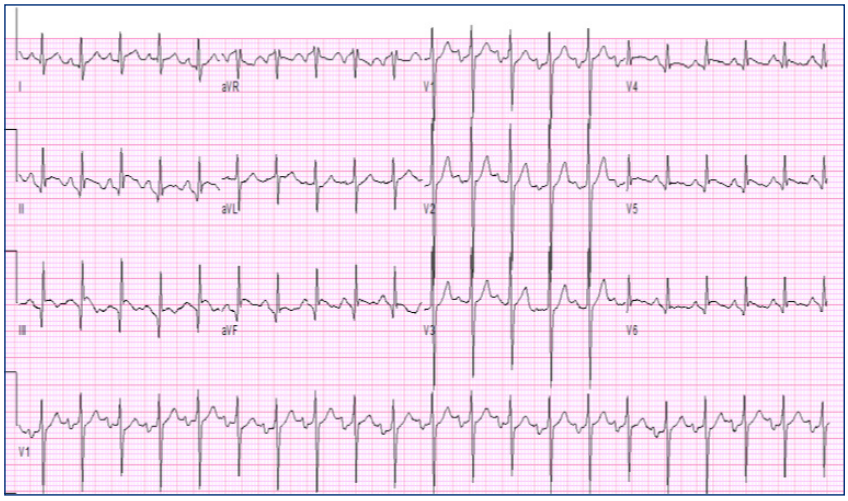


Image 3. Normal right coronary artery visualized in the LHC.

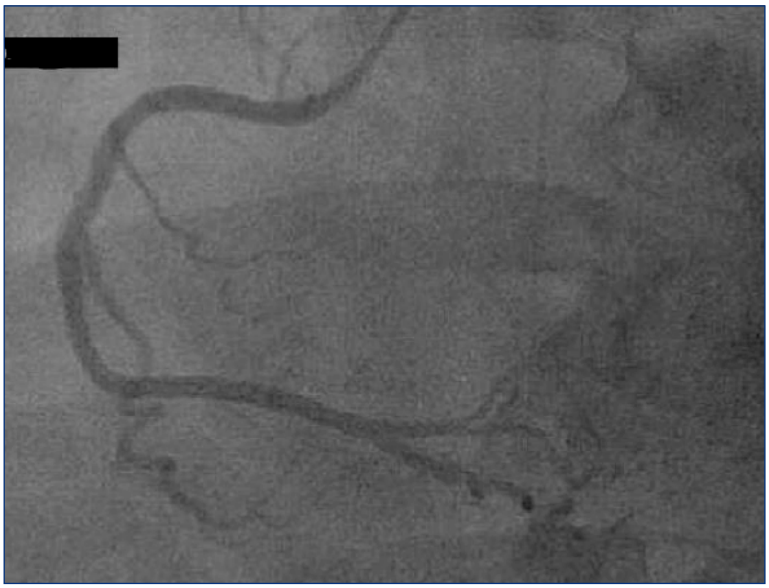
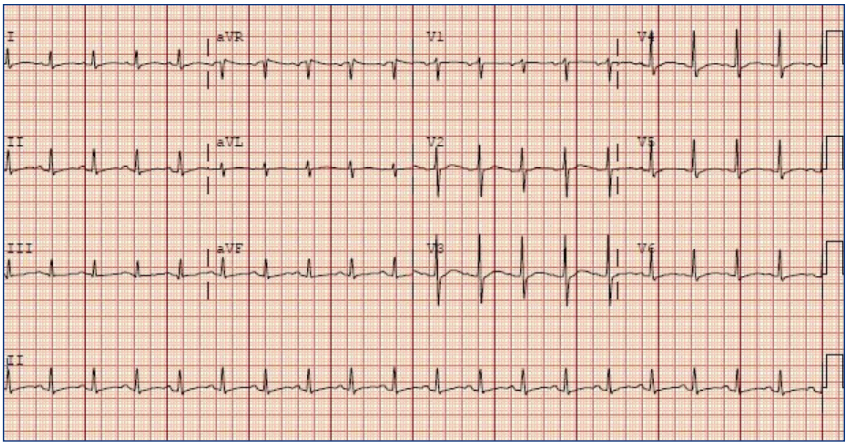


Image 4. Sinus tachycardia with resolution of the ST segment elevations in the inferior leads post decompression.



findings; this may be related to biliary cardiac reflex vs associated sepsis[11]. There is a paucity of data linking obstruction or dilation of the gut with ST segment elevations. Only one case was reported to have acute ST segment elevation intraoperatively due to small bowel obstruction[12].

There is no consensus on the mechanism by which these EKG changes occur when the actual pathology is below the diaphragm. As seen in **Image 1** our patient had a very impressive gastric dilation with increased air density secondary to ileus and as postulated by Chen et al. In our patient we believe the mechanism of ST elevations was due to the compression of the right coronary artery by the diaphragm under the intra-abdominal pressure caused by gastric air/ileus[9]. In all of this uncertainty the common characteristic that stands out in most of the case reports is: patients often have no coronary artery disease and the EKG changes resolve once the inciting factor is relieved. The cardiac enzymes are usually negative and invasive cardiac work-up including coronary angiography or echocardiogram reveals no abnormal findings, as in our patient.

This case illustrates the importance of understanding EKGs in context of the clinical scenario given that most of the reported cases with suspicious inferior MI suggest inferior wall compression related to abdominal pathology. In these settings, further evaluation should always be judicious. When vague symptomology and laboratory values contradict EKG findings, other differentials should be considered in order to avoid unnecessary therapies.

References

1. Durning SJ, Nasir JM, Sweet JM, Cation LJ. Chest pain and ST segment elevation attributable to cholecystitis: a case report and review of the literature. *Mil Med. 2006 Dec; 171(12):1255-8.*
2. Wang ZQ, Li YG. An unusual case of fulminant myocarditis closely mimicking ST-segment elevation myocardial infarction and presenting as refractory cardiogenic shock complicated by multiple life-threatening arrhythmias. Rev Port Cardiol. 2015 Dec;34(12):771.e1-5. doi: 10.1016/j.repc.2015.03.030. Epub 2015 Nov 27. English, Portuguese.
3. Chen A, Ren X. Aortic Dissection Manifesting as ST-Segment-Elevation Myocardial Infarction. Circulation. 2015 May 26;131 (21):e503-4. doi: 10.1161/CIRCULATIONAHA.114.015200.
4. Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med. 2003 Nov 27;349(22):2128-35.
5. Frais MA, Rodgers K. Dramatic electrocardiographic T-wave changes associated with gastric dilatation. Chest. 1990 Aug;98(2):489-90.
6. Patel N, Ariyarathenam A, Davies W, Harris A. Acute cholecystits leading to ischemic ECG changes in a patient with no underlying cardiac disease. JSLS. 2011 Jan-Mar;15(1):105-8. doi: 10.4293/108680811X13022985131534.
7. Pezzilli R, Barakat B, Billi P, Bertaccini B. Electrocardiographic abnormalities in acute pancreatitis. *Eur J Emerg Med. 1999 Mar; 6(1):27-9.*
8. Coppola G, Carita P, Corrado E, Borrelli A, Rotolo A, Guglielmo M, Nugara C, Ajello L, Santomauro M, Novo S. ST segment elevations: Always a marker of acute myocardial infarction? indian heart journal. 2013; 65(4), pp.412-423.
9. Chen Tsu-Yi, Ching-Hsiang Lin, Shih-Hung Tsai, Sy-Jou Chen. Pseudomyocardial infarction caused by expansion of colonic tube used for esophageal reconstruction. The American journal of the medical sciences. 2012;344(6): 499-500.
10. MH Shamma'a, GA Rubeiz. Acute pancreatitis with electrocardiographic findings of myocardial infarction. The American Journal of Medicine. 1962; 32(5):827–830.
11. ET Ryan, PH Pak, RW DeSanctis. Myocardial infarction mimicked by acute cholecystitis. Annals of Internal Medicine. 1992;116(3):218–220, 1992.
12. Patel K, Chang NL, Shulik O, DePasquale J, Shamoon F. Small Bowel Obstruction Mimicking Acute ST-Elevation Myocardial Infarction. Case reports in surgery, 2015.

Authors

Osama Siddique, MD, Providence VA Medical Center, The Warren Alpert Medical School, Brown University.

Somwail Rasla, MD, Providence VA Medical Center, The Warren Alpert Medical School, Brown University.

Seth Clark, MD, Providence VA Medical Center, The Warren Alpert Medical School, Brown University.

Aravind Kokkirala, MD, Providence VA Medical Center, The Warren Alpert Medical School, Brown University.

Correspondence

Osama Siddique, MD
Department of Medicine
Memorial Hospital of Rhode Island
111 Brewster St.
Pawtucket, RI, 02860
401-729-2258
Fax 401-729-2022
osama_siddique@brown.edu